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HIGH INTENSITY INTERVAL TRAINING VS STEADY STATE EXERCISE AND RELATION TO POST-EXERCISE HYPOTENSION

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HIGH INTENSITY INTERVAL TRAINING VS STEADY STATE AND THE RELATION TO POST-EXERCISE HYPOTENSION

By Brianna Roberts

We recommend acceptance of this thesis in partial fulfillment of the candidate's requirements for the degree of Master of Science, Clinical Exercise Physiology.

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ABSTRACT

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High intensity interval training has become popular to produce various physiological benefits. There is much controversy over what type of interval training has the greatest effects. Purpose: This study was designed to compare high intensity interval groups to examine levels of post-exercise hypotension (PEH). Methods: Young, healthy volunteers (N=55) performed 24 workout sessions on a stationary bike over an 8 week period. Subjects were randomly placed into three different exercise groups: Tabata, Meyer, and steady state. Tabata (N=21) performed 20 seconds at 170% power output (PO) of VO_2 max and 10 second rest for 8 bouts. Meyer (N=15) performed 30 seconds at 100% PO of VO_2 max, with 60 seconds active recovery, for a total of 20 minutes. Steady state (N=19) performed 20 minutes of exercise at 90% ventilatory threshold (VT). Blood pressures were measured once a week and multiple times during those sessions. Measurements of PEH were taken 30 minutes after exercise. Results: There were significant differences in systolic blood pressure (SBP) between pre- and post-exercise but no significant differences between the three exercise groups. Averaged over eight weeks, steady state had PEH of 9.1 $\pm$ 2.17, Meyer showed PEH of 8.3 $\pm$ 1.83 while Tabata had PEH of 9.1 $\pm$ 1.55. There was no significant difference in PEH between the weeks. Conclusions: Through this study, we have concluded that PEH occurs in SBP 30 minutes after exercise, regardless of exercise intensity. During the eight week training, PEH remained constant. Previous findings have concluded similar results, including studies involving blood pressure medication.

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INTRODUCTION

High intensity interval training (HIIT) has become a very popular form of exercise in recent years. This type of training is used in multiple places such as clinical settings, corporate training facilities, sports facilities and in recreational sport. There is much controversy over what level of HIIT has the greatest effects and whether it has a sufficiently good outcome to overbalance the possible risks of injury. For the purpose of our study, we used two HIIT workouts: one created by Katharina Meyer and the other by Izumi Tabata. Tabata, Irisawa, Kouzaki, Nishimura, Ogita, and Miyachi (1997) used a very high intensity of 170% power output (PO) at maximal oxygen consumption (VO_2 max), while Meyer, Samek, Schwaibold, Westbrook, Hajric, Lehmann, and Roskamm (1996) used patients working at 50% PO from a steep ramp test. This value is close to 100% of PPO of a normal ramp test. Using a very demanding regime of 20 seconds of work/10 seconds of recovery, Tabata et al. (1997) demonstrated increases in both aerobic power and anaerobic capacity following his training protocol, whereas a control group doing conventional aerobic training improved only aerobic power. The work of Tabata et al. (1997) extends work done by Gibala and McGee (2008) and Burgomaster, Howarth, Phillips, Rakobowchuk, MacDonald, McGee, and Gibala (2008) which demonstrated a unique potential to ultra-high intensity training.

Although conventional interval training has been used for many years in fitness and athletic settings (Knuttgen, Nordesjo, Ollander, and Saltin, 1973), Meyer et al. (1996) demonstrated that interval training could be extended into populations which had not

previously used interval training, widely, patients with cardiovascular disease. However, unlike Tabata et al. (1997), Meyer et al. (1996) used a relatively easier training program of 30 seconds work/60 seconds recovery. The recent interest in HIIT has raised the question of whether this is a more effective form of training, a more time efficient form of training, or just one of many fads that has captured the attention of the exercise community.

Beyond the classical increase in VO_2 max that is the cornerstone of the fitness community (Taylor, Buskirk and Henschel, 1955; Mitchell, Sproule and Chapman, 1958; Levine, 2008), another favorable outcome of exercise training, as a modulating factor for cardiac risk factors, is hypertension. There are predictable reductions in blood pressure (BP) after a period of regular exercise. There are also acute reductions in BP following a single exercise bout. The term post-exercise hypotension (PEH), coined in 1993 by Michael Kenney and Douglas Seals (Pescatello, Franklin, Fagard, Farquhar, Kelley, and Ray, 2004), was used to describe the exaggerated decrease in BP after an exercise bout. Post-exercise hypotension has been shown to last up to 22 hours (Pescatello et. al, 2004).

Potential mechanisms of PEH include a decrease in cardiac output (CO) and stroke volume (SV), although a decrease in total peripheral resistance (TPR) is thought to play the primary role in decreasing BP. The vasodilation, causing a decrease in TPR as well as a decrease in sympathetic nerve activation, contributes the most to PEH. Also, potentially contributing is the effect that exercise has on resetting baroreceptors. Afferent skeletal muscle receptors apparently play a large role in setting the baroreflex to keep pressure low in the vascular system (Halliwill, Buck, Lacewell, and Romero, 2013). Another aspect of vasodilation that has been studied is the production of nitric oxide

(NO). Nitric oxide is released from the endothelium of the vessels during exercise, especially more vigorous exercise to allow for blood flow to the working muscles (Halliwill, 2001).

Post-exercise hypotension has been shown to be affected by the use of different medications or supplements. Phosphodiesterase (PDE-5) inhibitors have been used to test the effects of PEH with these drugs in the system (Gehrke, Foster, Steenstra, Shatzer, Greany, Gibson, and Porcari, 2010 and Steenstra, Gehrke, Foster, Greany, Gibson, Shatzer, and Porcari, 2010). Angiotensin-converting enzymes (ACE) are found in the body and they assist with increasing BP. Angiotensin-converting enzyme inhibitors keep BP down (Beaulieu, Nadeau, Lacourciere, and Cleroux, 1993) and may work additively with PEH. The ingestion of the amino acid L-arginine, a NO precursor (Schuster-Decker, Foster, Porcari, and Maher, 2002), also lowers BP and increased the magnitude of PEH.

The purpose of this study was to compare two different HIIT programs with a steady-state training program (control). The Tabata group will have shorter bouts of training with very high intensity, while the Meyer group used longer bouts and a lower, but relatively sustainable overall intensity. To the degree that PEH is mediated by NO release, we predict that PEH will be greater in the Tabata group, intermediate in the Meyer group, and minimal in the steady-state control.

METHODS

The University of Wisconsin – La Crosse Institution Review Board for the Protection of Human Subjects approved the protocol for this study. The subjects provided written informed consent after both written and verbal descriptions of the protocol. Originally, 65 subjects were used for this study. Ten dropped out due to lack of interest or injury unrelated to this study. The remaining 55 healthy, sedentary young adults were recreationally active less than twice a week and were randomly assigned to their training groups. The Tabata group started with 24 students and lost three due to injury unrelated to the study or lack of interest, meaning 21 subjects completed the trial. The Meyer group started with 21 and lost six due to lack of interest or injury unrelated to the study, meaning 15 subjects completed the study. The control group (steady-state exercise) began with 20 subjects and lost one due to lack of interest; therefore, 19 students completed the study. Characteristics of the subjects are presented in Table 1.

Table 1. Descriptive Characteristics of Subjects (mean ± SD)

	Steady-State (19)	Tabata (21)	Meyer (15)
Age (yrs)			
Males	19.5 ± 1.38	20.3 ± 2.14	19.3 ± 1.26
Females	19.6 ± 2.94	19.5 ± 1.16	19.9 ± 2.77
Height (cm)			
Males	181.5 ± 8.94	174.6 ± 6.08	179.3 ± 10.69
Females	164.8 ± 4.87	168.6 ± 3.93	164.9 ± 4.85
Weight (kg)			
Males	94.3 ± 7.22	81.0 ± 13.85	76.4 ± 12.47
Females	68.6 ± 15.12	68.2 ± 14.04	71.9 ± 18.55

Each participant completed pre and post VO_2 max and Wingate tests to assess aerobic power, anaerobic power and anaerobic capacity. The initial intensity for each subject was calculated from these pre-test values. The training groups included the Tabata group (20 seconds at 170% PO at VO_2 max and 10 second rest for 8 bouts), a Meyer training group (30 seconds 100% PO at VO_2 max, with 60 seconds active recovery), and a steady-state training group (20 minutes at 90% VT). Each group had a five minute warm up and five minute cool down.

Training sessions for each group took place three times a week for eight weeks and BP was measured weekly. The workload was adjusted weekly to maintain the same relative intensity. The same researcher measured all BPs using the auscultatory method. Subjects were seated in an upright position during BP measurements, which occurred once a week, multiple times throughout the session. Blood pressure was taken prior to exercise, during the last minute of vigorous exercise, and 30 minutes after cool down was complete. Subjects were asked to sit quietly in the laboratory, and stay seated for the entire 30 minutes before the last BP measurement was taken. The subjects were asked to read, and not to engage in conversation during the post exercise recovery period.

Data were analyzed for differences between the exercise groups using change from pre-exercise to 30 minutes post-exercise for systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) using repeated measures ANOVA. Tukey's post hoc test was used to isolate differences between the means. Alpha was set at 0.05 to achieve statistical significance.

RESULTS

Changes in BP are shown in Figures 1-3 for SBP, DBP, and MAP, respectively. Systolic blood pressure decreased significantly 30 minutes post-exercise in all three groups; however, there was no significant difference between the three groups in PEH. There were no significant differences in the change in DBP and MAP between pre- and post-exercise measurements. We also assessed the difference in SBP change across the weeks of the study, but there was no statistical significant difference. The average decrease in SBP in week 1 was 12.6 $\pm$ 10.48 mm Hg (millimeters of mercury) while week 2 had a decrease of 7.2 $\pm$ 11.58 mm Hg. The following weeks had similar changes to week 2. There were no significant changes in DBP and MAP in week 1 compared with the remaining weeks. So in summary, PEH was evident for SBP, equally in all groups. In the first week of training, the effect was larger (10-15 mm Hg). Afterwards the magnitude of PEH was less (5-10 mm Hg).

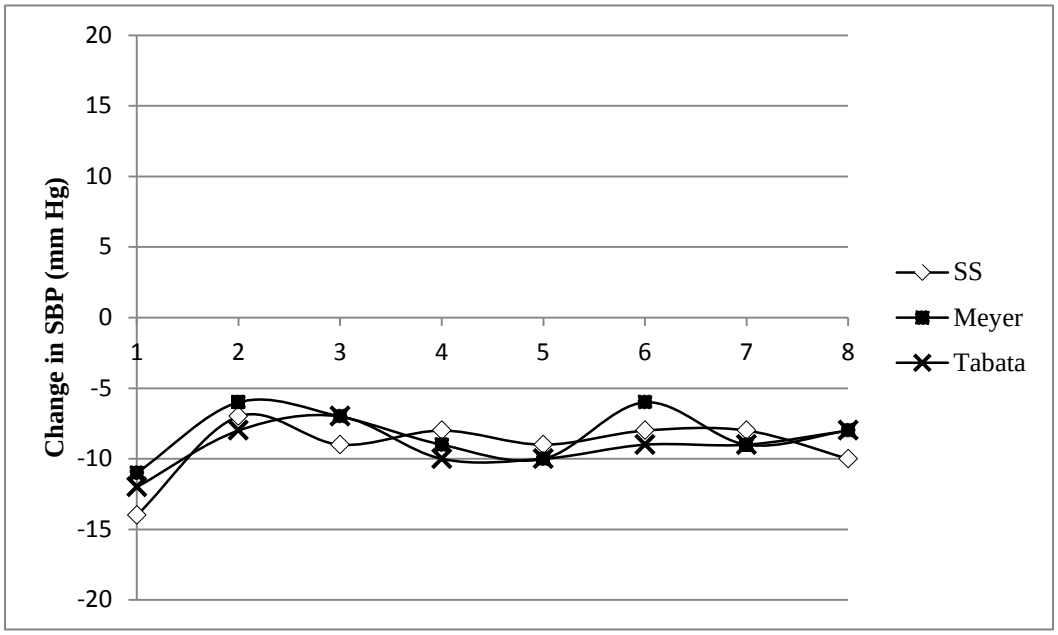

Figure 1. Systolic Blood Pressure Change over Weeks
Shows a change in SBP by weeks by group. The change between pre-exercise and post-exercise SBP was significant. The greater magnitude of change in week one was not significant from the rest of the weeks. The change between the groups was not significant.

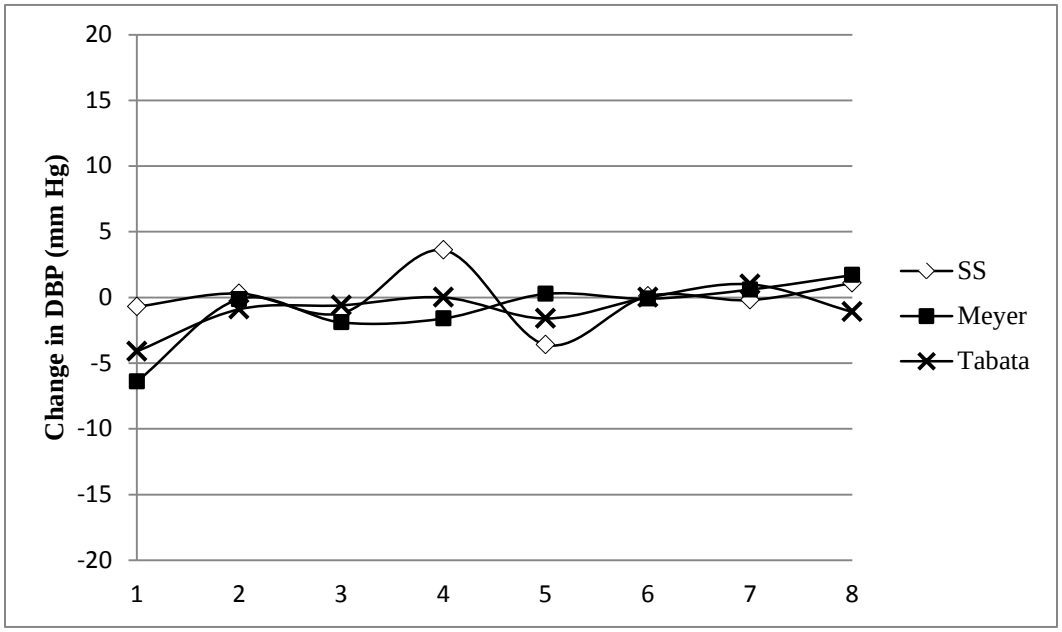

Figure 2. Diastolic Blood Pressure Change over Weeks
The change between pre-exercise and post-exercise in DBP. There was no significance in the difference from pre- to post-exercise DBP.

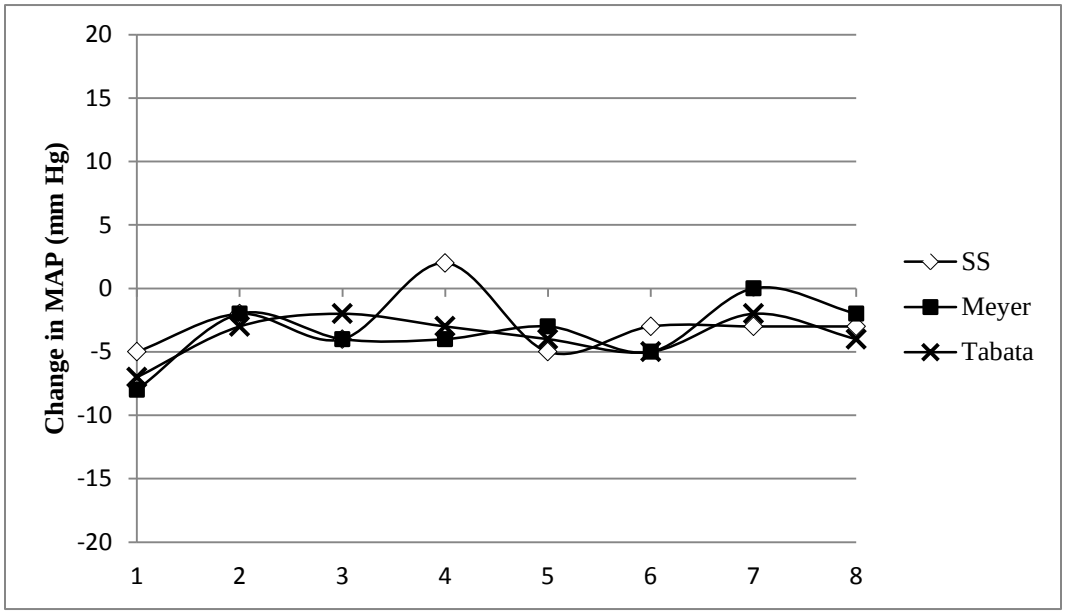

Figure 3. Mean Arterial Pressure Change over Weeks
Mean arterial pressure was calculated (DBP + 1/3[SBP-DBP]) and is portrayed in this graph. There was no significant change from pre-exercise to post-exercise or between exercise groups.

DISCUSSION

High intensity interval training is thought to have larger exercise benefits than conventional steady-state training. However, the results of this study demonstrated that PEH was not larger following more intense exercise. The three groups all showed significant decreases in post-exercise SBP, but the type of exercise training did not make a difference in PEH. In the steady-state group, SBP decreased by an average of 9.13 $\pm$ 2.17 mm Hg post-exercise throughout the eight weeks. The Meyer group decreased SBP by 8.25 $\pm$ 1.83 mm Hg while the Tabata group decreased 9.13 $\pm$ 1.55 mm Hg. Systolic blood pressure was the only measurement that showed a statistical significant reduction.

There are many mechanisms of PEH that could explain the results observed during this study. Pescatello et al. (2004) explains in the American College of Sports Medicine Position Stand on Exercise and Hypertension that "decreases in catecholamines and total peripheral resistance, improved insulin sensitivity, and alterations in vasodilators and vasoconstrictors are some of the explanations." Hypertension is defined at SBP >130 and DBP >85 as stated by Pescatello et al. (2004). Decreased TPR usually causes the most affects in PEH, but discovering why PEH occurs for so much time after exercise is still something to investigate. Mean arterial pressure was not significantly different in our results and is the product of CO and TPR. For the purpose of this study, MAP was calculated as DBP + (1/3 [SBP-DBP]). Cardiac output is the product of heart rate (HR) and SV. Both of these characteristics increase during exercise and take more time than TPR to return to baseline levels. Because CO does not immediately decrease

with the cessation of exercise, TPR has to play a larger role to keep BP down after exercise (Pescatello et al., 2004).

There are several mechanisms that can influence vasodilation and the decrease in BP. A major contributor to vasodilation is the release of NO from the endothelium (Halliwill, 2001). With chronic exercise and improved endothelial function, the vessels are able to release more NO to increase vasodilation, resulting in a decrease in TPR. With acute exercise, as shown in our research, the endothelium releases NO to allow blood flow to the tissues during and after exercise. The NO mediated vasodilation creates the immediate drop in BP after exercise, but other mechanisms help sustain BP, such as the baroreflex (Mach, Foster, Bricew, Mikat, and Porcari, 2005).

The baroreflex and sympathetic nervous system inhibition can also create a decrease in BP contributing to PEH. Not only does NO create a vasodilatory effect, but the inhibition of the sympathetic nervous system allows the vessels of non-working muscle groups to relax and vasodilate, creating an even lower TPR, hence a decrease BP (Halliwill et al., 2013). The baroreflex can play a part in sustaining PEH for as long as 22 hours (Pescatello et al., 2004). When exercise is completed, the baroreceptors are reset, and over-compensate causing the BP to return to a lower level. High BPs during exercise presumptively cause the baroreceptors to reset to a level lower than prior to exercise (Halliwill et al., 2013). Many of the mechanisms involved with PEH are unclear and not entirely known when it comes to intensity and type of exercise.

Studies have shown that hypertensive patients usually have greater PEH results than those who have blood pressure in normal ranges. Pescatello, Fargo, Leach, and Scherzer (1991) showed similar results to this in their study. They looked at ambulatory

BP in both hypertensive and normotensive subjects. Pescatello et. al (1991) found that hypertensive subjects produce the same amount of PEH whether they are performing at 40% VO_2 max or 70% VO_2 max. Those who were hypertensive were off their medication for this study and had significant decreases in SBP for up to 12 hours after exercise. They also demonstrated that BP stayed 9-11 mm Hg lower in hypertensive patients on days they exercised compared to days where they did not exercise. There were no significant changes in subjects with normal BP. This supports the conclusion that exercise can help lower and sustain low BP.

Medication can have a large effect on BP and its response to exercise. Two medications that are often used to treat high BP are diuretics and ACE inhibitors (Heran, Wong, Heran, and Wright, 2008). Heran et al. (2008) reported that ACE inhibitors without exercise lowered SBP by about 8 mm Hg. Our subjects had decreases in BP ranging from a low of 6 mm Hg to as high as 14 mm Hg post-exercise. This shows that exercise can have effects of comparable magnitude to medication relative to lowering BP. Beaulieu et al. (1993) found that ACE inhibitors decreased mean arterial pressure (MAP) similarly to the placebo group used in this study. The use of ACE inhibitors in hypertensive patients created 10 mm Hg more PEH 30 minutes after exercise. The difference between groups in this study was evident in reduced forearm vascular resistance (FVR). Total peripheral resistance and CO were affected equally but FVR was increased following the use of an ACE inhibitor. The assumption was made that ACE inhibitors cause increased vasodilation in other capillary beds in the body to maintain reduced MAP.

Other enhancing substances, such as drugs and supplements can affect PEH. In our laboratory, Schuster-Decker et al. (2002) demonstrated that the magnitude of PEH was enhanced for as long as 90 minutes after exercise if the exercise bout was preceded by consumption of a food product containing L-arginine, the amino acid precursor of NO. Schuster-Decker et al. (2002) found that L-arginine has a significant additive effect on PEH. Vitamin C and Vitamin E are also supplements that enhance NO synthesis, resulting in vasodilation and decreased BP. The amino acid was added to an exercising group and to a resting group, while a placebo was added to a separate resting and exercise group. Of the four groups, the subjects who were taking L-arginine had the greatest PEH 90 minutes after exercise. Blood pressure measurements were taken 30, 60, and 90 minutes after exercise; this allowed us to compare 30 minutes after exercise with our study. Those who ate the bar and performed 30 minutes of exercise saw a SBP change (-7 mm Hg) 30 minutes post-exercise. Those who did not perform exercise but ate the L-arginine bar also saw decreased SBP after 30 minutes of ingestion (-8 mm Hg). These results are corrected for the control because a fourth group (control) performed no exercise and did not eat the L-arginine bar but still saw a decrease in SBP of 5 mm Hg.

Studies in our laboratory have also been done using PDE-5 inhibitors, e.g. Viagra (Gehrke, 2010 and Steenstra, 2010). The destruction of NO is prevented by the work of PDE-5 inhibitors. By reducing the destruction of NO, the smooth muscle is able to fully relax to create vasodilation. This increase in vasodilation allows for a decrease in TPR which is thought to be the main contributor to PEH (Gehrke et al., 2010). Gehrke et al. (2010) studied middle aged men taking PDE-5 inhibitors during moderate to heavy exercise. They found that PDE-5 inhibitors, taken an hour before exercise, lowered the

initial resting BP but it did not contribute to a faster rate of BP decrease after the inhibitor was ingested. Post-exercise hypotension found after a 40 minute exercise bout was most likely influenced by the initial decrease in resting BP. Steenstra et al. (2010) found similar results; those taking the PDE-5 inhibitors experienced lower BP, but this seemed to be due to the initial decrease before exercise. In these studies, a few subjects who performed heavier exercise (e.g. greater NO release) had evidence of PEH, suggesting that some of the mechanism behind PEH is dependent on NO release.

There are data supporting our findings with PEH and intensity levels. The amount of work each group had done was comparable. Data from Jones, George, and Edwards (2007) and Liu, Thomas, Sasson, Banks, Busato, and Goodman (2012) support the concept that total work will create equal amounts of PEH. Jones et al. (2007) found that when two groups were exercising at different intensities but performing the same amount of work, the magnitude of PEH was approximately the same. Liu et al. (2012) also found similar results. Their study involved middle-age and younger aged men who performed two exercise bouts at separate occasions. One bout was at 60% VO_2 max while the other was at 80% VO_2 max. Liu et. al (2012) witnessed middle-aged men having greater PEH (-12 mm Hg with moderate intensity, -15 mm Hg with high intensity) than younger men (-1 mm Hg with moderate intensity, -5 mm Hg with high intensity). The conclusion was that older men had higher starting BP, which could be the reason for the larger magnitude of PEH. On the contrary, Smelker, Foster, Mahar, Martiniz, and Porcari (2004) found that higher intensity exercise produced greater PEH 30 minutes post-exercise. Their study included volunteers performing at 70, 80, 90, and 100% of ventilatory threshold (VT). Exercise at 100% VT was the only exercise group with significant PEH 30 minutes after

exercise. The group that exercised at low intensity (70% VT) was the only group who did not have significant decrease in SBP 120 minutes after exercise.

The duration of each training session may also have an effect on PEH. Mach et al. (2005) studied the effects of length of the exercise bout with volunteers who were performing at 80% VT. He found that exercise needed to last for at least 20 minutes for PEH to persist as long as 90 minutes post-exercise. This could explain why our Tabata group did not have a greater magnitude of PEH than the other two groups. Regardless of the higher intensity, the length of the Tabata workout was only four minutes. This may not have been a sufficient duration to see the effects of higher intensity exercise on PEH.

With interval training and high intensity training becoming more popular, it is important to understand which type of exercise has the best effects. Gibala and McGee (2008) found that interval training creates greater physiologic effects, such as increased VO_2 max, metabolism and other metabolic factors, but the question remains how much intensity is needed. Little, Safdar, Wilkin, Tarnopolsky, and Gibala (2010) found that low volume HIIT at 100% PO at VO_2 max for 60 seconds created similar results to those who were performing 'all-out' sprint Wingate bouts. Low volume HIIT is recommended for the general population where 'all-out' sprint intervals should be limited to athletes. These examples show the positive effects that HIIT has on the population, but also make it clear that the intensity needs to be specific to those who are performing the exercises. It suggests that there may be minimal values of both intensity and duration that are necessary to produce meaningful levels of PEH.

Studies have also been done with resistance training and PEH. In 2006, Rezk, Marrache, Tinucci, Mion, and Forjaz had subjects perform a number of exercises at 40%

one repetition max (1RM) and 80% 1RM. Both groups had significantly lower SBP but DBP was only lower in those participating in the 40% 1RM group**.** Keese, Farinatti, Pescatello, and Monteiro (2011) found that performing resistance training exercises was better for PEH if done with aerobic exercises. Resistance training exercises alone showed no significant change in PEH (Keese et al. 2011).

Multiple studies have been done on PEH with cardiac patients, who tend to be hypertensive, and on subjects who are normotensive. The results have varied. Forjaz, Cardoso, Rezk, Santaella, and Tinucci (2004) and Keese, Farinatti, Pescatello, Cuha, and Monteiro (2012) found that groups exercising at higher intensity experienced a larger and longer PEH. Jones, Taylor, Lewis, George, and Atkinson (2009) showed that interval training, compared to continuous training, produced greater levels of PEH.

The change in PEH in this study did not have any relationship with change in intensity, meaning that this change is not NO dependent under the circumstances of our study. This is consistent with Pescatello et al. (1991) with ambulatory BP measurements showing that BP remained low on days when exercise was performed. Exercise helps lower BP, about as much as BP medications such as ACE inhibitors, according to both our research and to the results of Heran et. al (2008) and Pescatello et. al (1991).

Figure 4 summarizes the PEH that occurred in the studies mentioned throughout this paper. The graph is organized with studies using drugs (ACE inhibitors and L-arginine) listed first, followed by other exercise studies, and then studies performed in our laboratory, ending with the results of the current study. As seen in the graph, BP medication can lower SBP by 5-10 mm Hg, even more with exercise. Hypertensive subjects have PEH of about 10 mm Hg while normotensive subjects have PEH of 5-10

mm Hg. The effect exercise type has on PEH is not large, and is not apparent at all in our study.

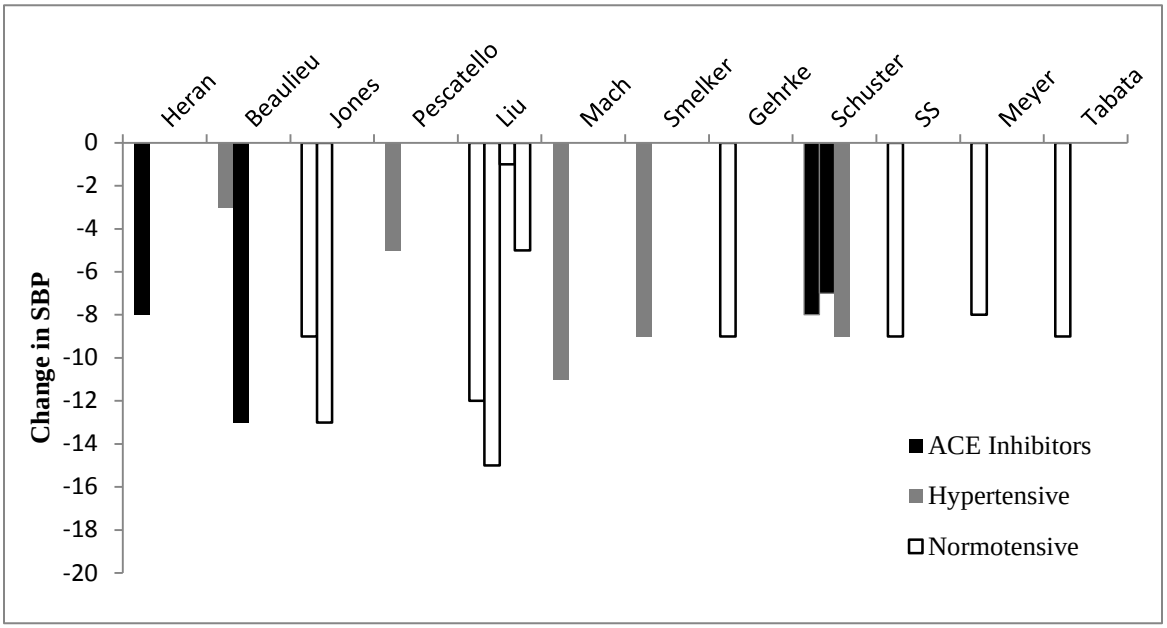

Figure 4. Summary Chart
Estimated PEH of multiple studies is relative to our recent study because PEH was collected 30-60 minutes post-exercise, and similar intensity levels were used.

Our subjects were normotensive so PEH may have been less evident than it would have been in hypertensive individuals. Replication of the general design of this study with hypertensive subjects would be of interest. Particularly since the results of this study did not show a progressive reduction in BP with continued training, it seems safe to assume that (just as with a drug) PEH is a relatively acute effect, which may profitably be studied in single exercise states.

In conclusion, in this study, the level of intensity had little or no effect on how much PEH was observed 30 minutes after exercise. Systolic blood pressure significantly

decreased in all three groups post-exercise while DBP and MAP did not change. There are several mechanisms that control PEH that could explain why intensity had minimal contribution to PEH. Increased vasodilation due to NO or sympathetic nervous system inhibition has a large impact on BP. However, neither the Tabata group nor the Meyer group (both of whom should have had higher levels of NO release) had more PEH than the control group. The baroreflex also affects TPR and could be another explanation of PEH. Potentially more prolonged programs might have produced a greater level of PEH.

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APPENDIX A

INFORMED CONSENT

Informed Consent for "High Intensity Interval Training vs
Steady State Exercise and the Relation to
Post-Exercise Hypotension"

Principle Investigator: **Brianna Roberts**
1118 Pine Street
La Crosse, WI 54601
(920) 210-9146

Purpose and Procedures:

- The purpose of this procedure is to see the relationship post-exercise hypotension has with different high intensity training intervals and steady state exercise.
- My involvement will consist of a pre- and post- VO2 max test and Wingate test, along with eight weeks of training four times a week under a specific exercise workout, where I was placed randomly. Each training session will last 30 minutes.
- During the eight weeks of training, I have been informed that my blood pressure and blood lactate will be taken once a week.
- I am aware that the testing will take place on a bike in the Human Performance lab at the University of Wisconsin – La Crosse

Potential Risks:

- I am aware I may experience fatigue and muscle soreness during and after exercise training bouts.
- I have been informed that in order to receive full compensation, extra credit points for HPR 105, I need to complete the entire eight week study along with pre and post exercise tests.
- The potential risk for life-threatening problems are minimal, but I am aware that I can stop the workout at anytime if I feel I can no longer continue.

Rights and Confidentiality:

- I have been informed that my results from this study will only be released to me and other researchers involved in this study. As for sharing results for publication, my results will be included as a group where my personal information will not be shared.
- I am aware that this study is voluntary and I am free to decline my participation at any time during this research study.

If there are any questions concerning this study, whether it be procedures, benefits, risks, or rights, they can be directed towards Brianna Roberts (920) 210-9146 or Dr. Carl Foster (cfoster@uwlax.edu) Questions concerning protection of human subjects may be directed towards the Institutional Review Board for the Protection of Human Subjects at UW – La Crosse at 608-785-8124 or irb@uwlax.edu.

Participant: _____
Date:_____

Researcher: _____
Date:_____

APPENDIX B

REVIEW OF LITERATURE

REVIEW OF LITERATURE

High Intensity Interval Training

Introduction

Multiple studies have been done regarding high intensity interval training (HIIT). The question still remains what mode of exercise is best, what level of intensity yields the best results, and the duration of the exercise bouts and resting time in between. Studies done by Izumi Tabata, Nishimura, Kouzaki, Hirai, Ogita, Miyachi, and Yamamoto (1996) have used a method of 20 seconds of 170% maximal oxygen consumption (VO_2 max) and 10 seconds of complete rest for 7 or 8 total bouts of exercise. It was determined that this high intensity workout created greater anaerobic effects and aerobic effects as well compared to continuous exercise. Tabata, Irisawa, Kouzaki, Nishimura, Ogita, and Miyachi (1997) continued his research by conducting a study using the same high intensity criteria (20:10 at 170% VO_2 max) and comparing to an exercise group performing at 200% VO_2 max with 30 seconds of activity and 2 minutes of rest. Despite the fact that the second group had done more total work, the 2 minutes of rest was too long to create the depletion of oxygen great enough to effect anaerobic capacity. It was here that the "Tabata" term was used for workouts that are still popular today.

Another study done by Helgerud, Hoydal, Wang, Karlsen, Berg, Bjerkaas, Simonsen, Helgesen, Hjorth, Bach, and Hoff (2007) resulted in higher VO_2 max. These subjects were exercising at an intensity of 90-95% maximal heart rate (HRmax); one

group at 15 X 15 seconds and another group 4 X 4 minutes. Both groups showed similar results.

Patients

Studies dealing with interval training have been done in cardiac rehabilitation patients dating back to 1963. Smodlaka (1963) had done a study observing heart rates (HR) of patients and found that more intervals created a lower resting HR. This helped with exercise prescription to see what intensity was used to keep the HR at 160 beats per minute, which at the time was sufficient for patients.

Guiraud, Juneau, Nigam, Gayda, Meyer, Mekary, Paillard, and Bosquet (2010) found similar results that more intervals meant better health outcomes when he conducted a study with coronary heart disease patients. Patients who were working at a level of 80% VO_2 max for 15 sec with 15 sec of complete rest had better results than those working for longer periods of time or those with an active recovery. This allowed for better patient safety and comfort.

Rognmo, Hetland, Helgerud, Hoff, and Slordahl (2004) confirmed that patients with coronary artery disease were able to increase their peak oxygen uptake (VO_2 peak) while working in intervals at a higher intensity of 80-90% VO_2 peak compared to a continuous exercise group at 50-60% VO_2 peak. The group performing the higher intensity performed a greater amount of work so the question arises if total work done has a greater affect on VO_2 peak and not just intensity.

Katharina Meyer has done various studies on patients and interval training. In 1990, Meyer, Lehmann, Sunder, Keul, and Weidemann conducted a study with patients who received bypass surgery and found that interval training with a minute of active rest

was better for patients than continuous training. Interval training allowed them to exercise for longer, and complete more work in a single session. In 1996, Meyer, Samek, Schwaibold, Westbrook, Hajric, Lehmann and Roskamm devised three different interval groups at 50% (30/60s) 70% (15/60s) and 80% (10/60s) power output PO at VO_2 max. Regardless of the intensity or duration of each exercise bout, the three groups showed similar health effects; therefore, it was advised that the lower intensity be used for patients for safety reasons. The following year, Meyer, Samek, Schwaibold, Westbrook, Hajric, Beneke, Lehmann, and Roskamm (1997) performed a study with patients with severe chronic heart failure. Again, it was found that interval training even after surgery helps increase aerobic capacity better than continuous exercise, even if the total amount of work done is significantly lower.

'All-out' Sprint Training

Burgomaster, Hughes, Heigenhauser, Bradwell, and Gibala (2005) performed a study observing 'all-out' sprint interval training (SIT). After performing 4-7 Wingate tests each session for a total of two weeks, this study had shown that SIT did not change anaerobic capacity mainly due to chronic fatigue, but it did have a positive effect on muscle oxidative potential and endurance capacity. These subjects were recreationally active volunteers and participated in exercise 2-3 times per week. Whyte, Gill, and Cathcart (2010) performed a similar study on overweight/obese men. After two weeks of 4-6 Wingates per session for six total sessions, the results showed an increase in Wingate power and VO_2 max including a number of other metabolic factors. It was stated here that "it is premature to recommend SIT in the form used here as a physical activity strategy to

the general population." This amount of workload may cause too much risk in certain populations.

Bayati, Farzad, Gharakhanlou, and Agha-Alinejad (2011) performed a study comparing 'low-volume high-intensity interval training to an "all-out" SIT.' The SIT group performed 3-5 cycling bouts for 30 seconds at an all-out intensity, with 4 minutes of recovery. The lower volume group performed 6-10 cycling bouts at 125% PO at VO_2 max with a 2 minute recovery. Both groups yielded similar results, and it was found that the intensity of the all-out group was a higher risk for the general population and but should be incorporated in the training program of athletes.

Total Work Done

There is no question that interval training rather than continuous training has a greater effect on a variety of health factors. Interval training can affect VO_2 max, increase muscle oxidative capacity, metabolism and multiple other metabolic factors (Gibala and McGee, 2008). The question remains exactly how high intensity needs to be to achieve these positive changes. Gibala et al. (2008) found that HIIT is a time-efficient way to stimulate these adaptations but in young healthy persons with a regular fitness routine. Seiler, Joranson, Olesen, and Hetleid (2013) did a study creating interval training groups. The group who performed 4 X 8 min intervals sessions at 90% HRmax had a greater VO_2 max than the 4 X 4 group at 95% HRmax. The total work done by these groups was the same, and yet the lower intensity achieved greater results. It was stated that this workload created a "manageable RPE" which allowed the subjects to maintain it for a longer period of time.

Little, Safdar, Wilkin, Tarnopolsky, and Gibala (2010) found similar results in a study that compared 'all-out' Wingate tests to low-volume HIIT. The low-volume HIIT group performed 60 seconds at 100% VO_2 peak with 75 seconds of recovery. This study showed that the low-volume HIIT group still was able to increase skeletal muscle mitochondrial capacity and improve exercise performance. Again, this type of exercise is recommended for general population.

Exercise Prescription

Prescribing levels of exercise can be difficult, especially amongst the general population. A collaborative article by Tschakert and Hofmann (2013) states that "interval exercise is a discontinuous mode of endurance exercise that is characterized by relatively short bouts of high-intensity workloads interspersed by periods of rest or low-intensity activity." It was also stated that intermittent exercise consists of five components: peak workload, duration, recovery load, recovery duration, and mean load, but the diversity available to prescribe exercise creates contradictory findings and opinions. Changing any of these five variables can be demanding on a patient and perhaps even dangerous. Prescribing exercise may have better and safer results if intensities are done using lactate or ventilation thresholds. Active recovery usually facilitates lactate oxidation in working muscles, which is why an active recovery may create lower physiological responses during HIIT compared to passive recovery.

Post-Exercise Hypotension

Mechanisms

Many of the mechanisms that are involved in producing post-exercise hypotension (PEH) are unclear but recent research has been able to produce some clarity. The term PEH was first coined in 1993 (Pescatello, Franklin, Fagard, Farquhar, Kelley, and Ray 2004). Pescatello et al. (2004) explains in the American College of Sports Medicine Position Stand on Exercise and Hypertension that "decreases in catecholamines and total peripheral resistance, improved insulin sensitivity, and alterations in vasodilators and vasoconstrictors are some of the explanations." PEH has also been said to last up to 22 hours. Hypertension can cause a decrease in the endothelial function. Part of the role of vascular endothelium is the release of nitric oxide (NO). Nitric oxide plays an important role in vasodilation which decreases blood pressure (BP). Exercise increases the production of NO and can decrease total peripheral resistance (TPR) which also plays a very large role in PEH. (Halliwill, 2001). Total peripheral resistance plays a larger role in PEH than cardiac output (CO). The product of CO and HR is mean arterial pressure (MAP). The increased HR during exercise does not always allow for a decrease in CO immediately after exercise, so in order to keep (MAP) down after exercise, TPR needs to play a larger role (MAP = CO x TPR) (Pescatello et al., 2004).

Other mechanisms of PEH that need to be considered are sympathetic nervous system inhibition and an adjustment in the baroreflex (Kammermeier, 2004 and Mach, Foster, Brice, Mikat, Porcari, 2005 and Halliwill, Buck, Lacewell, Romero, 2013). Sustained vasodilation occurs when the sympathetic nervous system is no longer activated which occurs acutely after exercise has ceased. During exercise, the

sympathetic nervous system contracts vessels in the non-working muscles, so the dilation of these muscles add to the sustained vasodilation potentially used in PEH. The baroreflex is another explanation of PEH. It is a homeostatic mechanism used to keep BP at a homeostatic level. When exercise is completed, the baroreceptors are reset, causing the baroreflex to decrease to a lower level, causing a lower BP which in this instance is PEH (Halliwill et al., 2013). Nitric oxide creates the immediate drop in BP while the baroreflex help sustain it (Mach et al., 2005).

Patients: Hypertensive

Post-exercise hypotension has been looked at mainly in hypertensive patients. According to Kessler, Sisson, and Short (2012) those who are diagnosed with hypertension and not going through treatment will benefit from HIIT. Studies did show that those patients who were already receiving treatment for hypertension did not have significant changes in BP through aerobic intensity exercise. However, SIT did create a significant change in systolic blood pressure (SBP).

Hypertension is explained as SBP >140 and diastolic blood pressure (DBP) >90 (Dlin, Nora, Silverberg, Bar-Or, 1982). Dlin et al. (1982) showed that hypertension achieved during exercise was an indicator of future hypertension. The levels they used were higher for exercise (SBP >200 and DBP>90), and found that after 5.8 years, a significant number of subjects were found to have hypertension.

Highest Intensity and Duration

When looking at PEH, the duration of the exercises and the intensity used needs to be taken into consideration. Mach et al. (2005) created a study used to look at the duration of exercise and compare PEH. He found that with mildly hypertensive subjects,

exercise at an intensity of 80% ventilatory threshold (VT) need to be longer than 20 minutes in order to sustain PEH, for up to 90 minutes post-exercise. Jones, Taylor, Lewis, George, and Atkinson (2009) looked at intervals and broke up a 30 minute steady state exercise at 70% VO_2 max into three sessions of ten minutes. PEH was found to be lower in the interval group compared to the continuous exercise group. Nybo et al. (2010) believed that a reduction in SBP after aerobic training is not directly correlated with VO_2 max results but still found that lower intensities had a lower impact on VO_2 max than moderate-intensity training.

Liu, Thomas, Sasson, Banks, Busato, and Goodman (2012) also found that groups working at 60% VO_2 max had similar PEH results as those working at 80% VO_2 max. This study was done in middle-aged men and younger men. Both groups performed exercises at each intensity but it was shown that PEH was lower in older men. This was most likely due to the lower baseline BP. A study done by Cornelissen, Verheyden, Aubert, and Fagard (2010) used men over the age of 55 and they performed a crossover study where subjects were sedentary, worked out for 10 wks at 33% VO_2 max and then worked at an intensity of 66% VO_2 max. No significant changes in PEH were found amongst the groups. Pescatello, Fargo, Leach and Scherzer (1991) conducted a study involving ambulatory BP. This cross over study was done with participants in which half were normotensive and half were hypertensive. One workout consisted of 40% VO_2 max while the other was at 70% VO_2 max. This study found that the intensity of the exercise did not affect PEH and that hypertensive participants had lower SBP throughout the day than on days where they did not exercise. Mean arterial pressure, SBP, and DPB all lowered in hypertensive participants up to 12 hours after exercise. Nothing was found in

the normotensive group. On the contrary, Molmen-Hansen, Stolen, Tjonna, Aamot, Ekeberg, Tyldum, Wisloff, Inful and Stoylen (2012) showed that working at an intensity of 90% VO_2 max had a greater PEH and for a longer period of time than groups working at only 60% VO_2 max. Similarily, Keese, Farinatti, Pescatello, Cuha, and Monteiro (2012) found that cycling bouts of various intensities created a greater and longer sustaining PEH at the highest intensity, which was 80% VO_2 max. Another study done at the University of Wisconsin – La Crosse by Smelker, Foster, Mahar, Martiniz, and Porcari (2004) showed that significant changes in PEH were found after the highest intensities which were 90% VT and 100% VT, and PEH was sustained for a longer period of time. Forjaz, Cardoso, Rezk, Santaella, and Tinucci (2004) also found that greater cycling intensities of only 75% VO_2 max were created a greater PEH and a longer lasting low BP.

Resistance Training

Most research on PEH has been done on aerobic training groups, and few look at resistance training groups. Keese, Farinatti, Pescatello, and Monteiro (2011) found that subjects who were doing resistance training and aerobic activity had similar effects on PEH two hours after exercise to those doing just aerobic activity and had greater effects than those just performing resistance training exercises.

Rezk, Marrache, Tinucci, Mion, and Forjaz (2006) found contradicting results that highest intensity creates the greatest PEH. He compared two groups performing resistance training exercises; one group performed at 40% one rep max (1RM) and the other group was 80% 1RM. SBP decreased similarly in both groups but DBP decreased more in the group performing at 40% 1RM.

Total Work Done

When it comes to total work done, results can vary and research is still needed to confirm an idea. Jones, George, and Edwards (2007) found that two interval groups performed the same amount of work, but one group performed at a lower intensity and results for PEH between the two were similar. A study done at the University of Wisconsin – La Crosse by Kammermeier (2004) found that interval workloads had no greater effects on PEH compared to a steady state group with the same amount of work done.

Medications

There are many medications that help with controlling BP, including Angiotensin-converting enzyme (ACE) inhibitors (Heran, Wong, Heran, and Wright, 2008). Heran et al. (2008) found that these medications, regardless of dose, created a decrease in SBP of 6 to 9 mm Hg. Medications and supplements can play a critical role in PEH, including phosphodiesterase inhibitors (PDE-5 inhibitors), ACE inhibitors, and the amino acid L-arginine. Studies at the University of Wisconsin – La Crosse have been done on PDE-5 inhibitors, more specifically, sildenafil (Viagra). Phosphodiesterase inhibitors are used to create more vasodilation in the body by eliminating the destruction of NO. By reducing the destruction of NO, the smooth muscle of the vessels is able to relax to create vasodilation. This increase in vasodilation allows for a decrease in TPR which is a main contributor to PEH. Steenstra, Gehrke, Foster, Greany, Gibson, Shatzer, and Porcari (2010) and Gehrke, Foster, Steenstra, Shatzer, Greany, Gibson, and Porcari (2010) performed studies on middle aged men taking Viagra. Gehrke et al. (2010) found that PDE-5 inhibitors lowered the initial BP that was taken at rest but it did not continue to

decrease at any faster rate after the inhibitor was ingested. The PEH found after the 40 minute exercise bout was most likely influenced by the initial decrease in resting BP. Steenstra et al. (2010) found similar results; he found those taking the PDE-5 inhibitors were experiencing lower BP, but this could be due to the initial decrease before exercise. The use of the drug Viagra was used for safety purposes. Viagra is an acute-acting drug compared to tadalafil (Cialis) which is a long-term PDE-5 inhibitor. Performing either of these studies with Cialis could have caused a greater chance of risk by decreasing PEH to harmful levels (Gehrke et al., 2010).

Post-exercise hypotension has been looked at after the use of different medications or supplements. As stated previously, PDE-5 inhibitors have been used to test the effects of PEH with these drugs in the system. Angiotensin-converting enzyme inhibitors have shown to help keep PEH down (Beaulieu, Nadeau, Lacourciere, and Cleroux, 1993) and the ingestion of the amino acid L-arginine (Schuster-Decker, Foster, Porcari, and Maher, 2002) is used to improve the production of NO. Angiotensin-converting enzymes are found in the body and they assist with increasing BP. Beaulieu et al. (1993) found that ACE inhibitors decreased MAP similarly to the placebo group used in this study. The difference between groups in this study was in the forearm vascular resistance (FVR). Total peripheral resistance and CO were affected equally but FVR was increased during the use of an ACE inhibitor. The assumption was made that ACE inhibitors caused increased vasodilation in other capillary beds in the body to maintain a MAP similar to the placebo group. Schuster-Decker et al. (2002) found that L-arginine has a significant additive effect on PEH. L-arginine is an amino acid that has a precursor effect on NO. Vitamin C and Vitamin E are also supplements that enhance NO synthesis,

in order to create vasodilation and inhibit platelet adhesion. The amino acid was added to an exercising group and to a resting group, while a placebo was added to a separate resting and exercise group. Of the four groups, the subjects who were taking L-arginine and exercise had a greater PEH.

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